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RUNNING HEAD: PSYCHOPATHOLOGY IN CHILDREN OF EATING DISORDERED MOTHERS

Abstract

Objective: There is evidence that parental psychiatric disorders are associated with offspring psychiatric disorder. Very few small studies have investigated the effect of maternal eating disorders on offspring psychopathology throughout childhood and early adolescence. We aimed to investigate psychiatric disorders at age 7, 10 and 13 years in offspring of women with eating disorders prior to pregnancy and investigate the relative contribution of other psychiatric disorders.

Method: Women (N=12,035) from a large population-based longitudinal cohort: the Avon Longitudinal Study of Parents and Children (ALSPAC). A brief pre-pregnancy psychiatric history was obtained at enrollment to determine exposure. Offspring psychiatric disorder was measured using the Developmental and Well-being Assessment at ages 7, 10 and 13.

Results: Maternal eating disorders were associated with a psychiatric diagnosis in the offspring at age 7 and 10, particularly emotional disorders (Odds ratio=1.9, 95%CI: 1.1-2.8). Maternal psychiatric disorders other than eating disorders predicted psychiatric diagnoses across ages, and acted in an additive fashion with maternal eating disorders.

Discussion: Maternal eating disorders together with comorbid psychopathology increase risk for psychiatric disorders in childhood and early adolescence, in particular for emotional disorders. This has important implications for prevention and future research.

The effects of maternal eating disorders on offspring childhood and early adolescent psychiatric disorders

Introduction

There is good evidence that parental mental ill-health is associated with psychopathology in the offspring.[1-3] In particular recent evidence from large population-based studies has highlighted an increased prevalence of psychiatric disorders in offspring of affected parents.[2,3] This evidence suggests that parental psychiatric disorders are associated with offspring disorder; moreover parental psychiatric disorder predicts a range of psychiatric disorders in the offspring, with little disorder specificity.[3] Unfortunately eating disorders (ED) were not investigated in the two large population-based studies above.[2,3]

Anorexia Nervosa (AN), Bulimia Nervosa (BN) and Eating disorders not otherwise specified (EDNOS) affect about 10% of women in reproductive age.[4] Although our understanding of the aetiology of ED is still limited,[5] there is evidence that both genetic and environmental factors contribute to the risk for ED[5] and that ED aggregate in families[6]. ED have a high level of comorbidity, especially with emotional disorders (both AN and BN)[7] and addictions (BN).[7] Two very small cross-sectional studies (on 19 and 27 children) from clinical samples found high levels of psychopathology in children of mothers with ED.[8,9] A more recent longitudinal follow-up of 33 ten-year-old children of ED mothers suggested a small effect of maternal ED on child emotional problems.[10]

Given the lack of research on the effect of maternal ED on offspring psychiatric disorders, and the possible role of general maternal psychopathology, we set out to investigate this in a longitudinal prospective study. In particular we aimed to: 1. Investigate the association of maternal lifetime ED with offspring psychiatric disorders

at three time-points in late childhood and early adolescence; and 2. Study the relative contribution of maternal co-occurring lifetime other psychiatric disorders.

Methods

Sample

The Avon Longitudinal Study of Parents and Children (ALSPAC) is a longitudinal, population-based, extensive prospective study of women and their children, set up in the '90s to investigate the effects of environment, genetics and other factors on child health and development.[11] All pregnant women living in the geographical area of Avon, UK, who were expected to deliver their baby between 1st April 1991 and 31st December 1992 were invited to take part in the study. More details can be found on http://www.bristol.ac.uk/alspac/. Children from 14,541 pregnancies were initially enrolled; of these 13,988 children were alive at 1 year. In the current analyses we excluded twins (n=199) and offspring of women who did not provide data on exposure, i.e. had not answered the questionnaire sent at approximately 12 weeks gestation enquiring about lifetime psychopathology (n=1,646). To avoid non-independence of the participants we also included only one offspring per mother, selected at random, if more than one contributed to the study (leading to excluding n=108). A total of 12,035 mother-offspring pairs were therefore included.

Outcomes:

The Developmental and well-being assessment (DAWBA) is a well-validated instrument for parental, teacher and young people completion, used across the world, that generates ICD-10 and DSM-IV diagnoses algorithmically.[12] The DAWBA is a semi-structured interview and has separate sections covering individual emotional, behavioural and hyperactivity disorders. The version used for this study was sent to parents and teachers (at age 7 only) as a paper questionnaire; ICD-10 and DSM-IV diagnoses were generated using a computer

algorithm (this method has been validated and shown to be useful in large epidemiological studies where clinician-rated diagnoses would be extremely difficult to obtain).[13]

The DAWBA was sent to parents at 3 time-points: when the child was aged 7, 10 and 13 years. At age 7 and 10 years 10,962 mothers and at age 13 years 10,614 mothers were sent questionnaires, with response rates of 58.8% at age 7, 55.9% at age 10, and 56.0% at age 13.[14]

Teachers were also asked to fill in the DAWBA when the child was aged 7; data were available on 5,476 children from this assessment.

Any Psychiatric disorder, emotional and behavioural disorders

Computer-generated diagnoses of any psychiatric disorder including emotional, behavioural disorders and attention deficit/hyperactivity disorder (ADHD) were obtained from the DAWBA at each time-point. The particular outcomes investigated were: any ICD-10 or DSM-IV disorder; any emotional disorder (ICD-10 or DSM-IV)- which included any anxiety disorder (ICD-10 or DSM-IV) and any depressive disorder (ICD-10 or DSM-IV); any behavioural disorder (ICD-10 or DSM-IV conduct disorder and/or oppositional defiant disorder) and any ADHD (DSM-IV). In addition specific anxiety disorders diagnoses (separation anxiety, specific phobia, social phobia, obsessive compulsive disorder, generalized anxiety disorder) were also used for descriptive purposes.

At age 7 diagnoses of any disorder, behavioural disorder and ADHD were generated using both parental and teacher report using the *or* rule (as per[13]). Teachers are deemed to be better reporters of behavioural and hyperactivity symptoms. In contrast emotional disorders diagnoses were obtained from

parental data only (for details see[13]). Computer-generated diagnoses using the parent rated paper or computerised DAWBA (and multi-informant DAWBA) have been shown to have moderate to high agreement (between 0.55 and 0.71) with clinician-rated multi-informant diagnoses of any disorder, emotional and behavioural disorders[13].

In sensitivity analyses, as an additional validator of psychopathology obtained via parental report only, we investigated parental and teacher report on the Strengths and Difficulties questionnaire (SDQ)[18] emotional disorders subscale score collected at age 7 from parents and teachers. This information was available on 3,886 children. The SDQ is a widely used instrument to assess childhood psychopathology translated into more than 40 languages. It has been validated against several other well-established instruments assessing childhood psychopathology.

Exposure:

Maternal history of ED pre-pregnancy was determined using questions administered as part of a questionnaire at 12 weeks gestation: all women were asked whether they had a recent or past history of AN, and whether they had a recent or past history of BN. In total 3.7% of women reported either or both disorders; 166 reported lifetime AN, 194 bulimia nervosa BN, and 81 both (see[14] for details). The latter group (AN+BN) was kept as a separate exposure group, given prior evidence in this same sample of higher prevalence of ED behaviours and low BMI.[14] Self-reported ED was validated in this sample using the shape and weight concern subscales of the EDE-Q and information on purging[15]. Moreover, self-report of AN and /or BN was recently shown to be highly be sensitive and specific: in a population-based study of pregnant women we found

that self-reported lifetime AN had a sensitivity of 100% and specificity of 96%; self-reported BN had a sensitivity of 94% and specificity of 81%.[16]

Additional predictor:

At 12 weeks gestation women were also asked about recent or past history of: severe depression, schizophrenia, alcoholism and other psychiatric disorders (n=1,301, 10.8% of the total sample). This information was combined into a variable indicating presence of any pre-pregnancy psychopathology other than ED: *other psychopathology*. Multiple answers were possible, therefore women who reported an ED could also report other psychopathology.

Covariates:

-maternal age, parity at birth of the offspring, maternal education obtained at recruitment or during pregnancy;

-child ethnicity obtained at birth.

Statistical analyses:

All outcomes were binary and were analysed using logistic regression models fitted separately at age 7, 10 and 13 years.

A sequential approach to the analyses was used: univariable models included firstly maternal ED (all ED) and secondly specific maternal ED (AN, BN, AN+BN) as the exposures of interest; the variable maternal "other psychopathology" was included in multivariable analyses in a second step, to obtain adjusted odds ratios. Thirdly, all effects were also controlled for confounders: namely maternal education, parity, child ethnicity, and child gender.

Because of missing data on maternal education, parity, and child ethnicity multiple imputation by chained equation with 10 imputation sets was implemented in Stata 12 (Stata Corp, 2011) assuming missing at random (MAR).[17] All predictors and outcome variables were used in the imputation model.

Results obtained after imputation were similar to those found when analyzing the complete records only; therefore results obtained from imputation models are reported throughout for simplicity.

To assess whether maternal other psychopathology affected the outcome independently or synergistically (on the log-odds scale) with the main exposure - maternal ED- maternal other psychopathology was included in the model as a dummy indicator with its interaction term with maternal ED also included.

To determine longitudinal effects of exposure on the outcomes we fitted a longitudinal logistic random effects model for cases with complete data only. This assumes (under the MAR assumption) that missing outcomes at later ages depend only on the observed outcomes, and the other variables included in the analyses (in this case the exposure and the confounders).

To assess whether the effect of the main exposure (maternal ED) on the outcomes (any psychiatric and emotional disorders) changed over the offspring age we fitted longitudinal random effects logistic regression models that included interaction terms between the exposure and dummy indicators of the ages when the outcome was recorded beyond baseline (i. e. age 10 and 13).

Attrition

Sample sizes were different at different ages. At age 7 data were available on 9,443 children (78.6% of the original available sample of mothers) on behavioral disorders and any psychiatric diagnoses (as above, due to the fact that these data incorporated teacher report as detailed in[13]); for emotional disorders data were available on 7,757 children (64.4% of the original sample).

At age 10 data were available on 7,069 (58.7% of the original sample) children. At age 13 6,438 children had data on the outcomes (53.5% of the original sample).

There was no evidence of selective attrition in relation to maternal ED exposure. However having a psychiatric diagnosis at age 7 predicted attrition at age 10 (OR=1.4, 95%CI: 1.1-1.7, $p<0.001$) and at age 13 (OR=1.7, 95%CI: 1.4-1.9, $p<0.001$); having a diagnosis at age 10 predicted attrition at age 13 (OR=1.8, 95%CI: 1.5-2.3, $p<0.001$).

All analyses were carried out in Stata 12. All statistical tests presented are two sided, with a $p<0.05$ used to define significance.

Ethics

The study was approved by the ALSPAC Law and Ethics committee and the Local Research Ethics Committees. All women gave informed consent at enrolment in the study.

Results

Socio-demographic data

Children of mothers with ED did not differ from those of non-ED mothers in relation to gender and ethnicity. Mothers with ED were more likely to be educated to A-level or degree level and to have a history of another psychiatric disorder prior to pregnancy compared to non-ED mothers (34.0% vs. 8.8%); amongst specific ED, women with AN+BN had the highest level of other psychopathology (53.2%) (see Table 1).

Maternal ED and offspring psychopathology at age 7

At age 7 any maternal ED predicted offspring DSM-IV or ICD-10 disorder in unadjusted analyses (OR=1.7, 95%CI: 1.2-2.4, $p \leq 0.01$), adjusting for maternal other psychopathology reduced the association, which become non-significant (see Table 2).

The strength of the association was similar across maternal ED type, although only the association between maternal BN and offspring disorder reached statistical significance in crude analyses.

Maternal ED strongly predicted emotional disorders at age 7 (OR=2.7, 95%CI: 1.6-4.7, $p \leq 0.001$); this effect decreased slightly after accounting for maternal other psychopathology (OR=2.2, 95%CI: 1.5-3.3, $p \leq 0.01$) and following inclusion of covariates.

The prevalence of offspring specific disorders are provided in Table 4.

Maternal ED types were differentially associated with offspring emotional disorder: a strong association was present for maternal AN and AN+BN (respectively crude OR=3.6, 95%CI: 1.6-7.9, OR=5.2, 95%CI: 2.0-13.2), with associations persisting in multivariable analyses (Table 2). Maternal BN was not associated with offspring emotional disorders. A very similar pattern emerged for offspring anxiety disorders: a strong association with maternal ED (AN and AN+BN), that reduced in strength when accounting for maternal co-occurring psychiatric disorders but remained statistically significant (Table 2).

Maternal ED were not associated with offspring behavioral disorders or ADHD at age 7.

Maternal ED and offspring psychopathology at age 10

At age 10 maternal ED predicted offspring DSM-IV or ICD-10 disorder in crude and adjusted analyses (crude and adjusted OR=1.7, 95%CI: 1.1-2.6, $p \leq 0.01$). Offspring of mothers with all ED types had higher odds of clinical disorders, with variable strength (two-fold higher odds) and the effects of maternal AN retaining statistical significance in multivariable analyses (Table 2).

Maternal ED were strongly associated with emotional and anxiety disorders (respectively crude OR=2.4, 95%CI: 1.4-4.1, $p \leq 0.01$ and OR=2.7, 95%CI: 1.5-4.9, $p \leq 0.01$), even when accounting for maternal other psychopathology. As for age 7 maternal AN and AN+BN were strongly associated with emotional and anxiety disorders at age 10, although, after adjusting for

maternal other psychopathology and covariates, maternal AN+BN only predicted offspring anxiety disorders.

At age 10 there was some indication of an increased risk of behavioral disorders in offspring of women with ED, despite the small numbers (Table 4) resulting in wide confidence intervals (Table 2).

No associations were found between maternal ED and ADHD.

Maternal ED and offspring psychopathology at age 13

At age 13 the prevalence of psychiatric disorder in offspring of women with ED was lower than in late childhood (Table 2). Although exposed children had increased odds of disorder, the strength of the association was lower compared to earlier waves, possibly due to attrition. Results only changed slightly when accounting for maternal other psychopathology.

Maternal AN+BN was associated with offspring disorder in crude analyses (OR=2.9, 95%CI: 1.2-6.8, $p \leq 0.05$), but less so once accounting for co-occurring psychiatric disorder (OR=1.9, 95%CI: 0.8-4.6, NS) (Table 3).

Crude positive associations between maternal ED and offspring emotional and anxiety disorders decreased in strength compared to previous time-points and once accounting for maternal other psychiatric disorders (Table 3). However, offspring emotional and anxiety disorders were associated with maternal AN+BN (emotional disorders: crude OR=5.3, 95%CI: 1.9-15.0, $p \leq 0.01$; anxiety disorders: crude OR=5.5, 95%CI: 1.7-18.2, $p \leq 0.01$). Both associations became less strong when accounting for maternal comorbid psychiatric disorders albeit remaining high and statistically significant (Table 3).

Offspring behavioral disorders and ADHD were not associated with maternal ED.

Maternal other pre-pregnancy psychopathology

Maternal other pre-pregnancy psychopathology was highly associated with any clinical offspring disorder (OR=2.2, 95%CI: 1.7-2.7, p≤0.0001) across all ages and emotional and behavioral disorders in univariable analyses, although associations decreased in strength in multivariable analyses (Table S2).

Sensitivity Analyses

In order to determine the possible effect of attrition on our findings we ran a series of sensitivity analyses.

To account for the missing outcomes at some waves of data collection under the missing at random assumption[17] we fitted a random intercept logistic regression model. This allows capturing individual trajectories in the odds of the offspring outcome via a random intercept.

Given the observed pattern of a decreasing strength of association between maternal ED and offspring outcomes (any psychiatric disorders and emotional disorders) and selective attrition of children with psychopathology at earlier waves, we carried out a series of sensitivity analyses to determine whether the decreasing strength of associations over time might be due to attrition.

Firstly we replaced missing values at age 10 (and 13) with the last observed outcome at age 7 (or 10) (scenario 1); secondly we replaced missing

values at age 10 (and 13) with the opposite of the value observed at the previous wave (scenario 2). Analyses were then carried out separately at each age.

At age 10 maternal ED were still strongly associated with offspring psychiatric disorders under both scenarios (scenario 1: adjusted OR=1.7, 95%CI: 1.2-2.3, scenario 2: OR=1.7, 95%CI: 1.1-2.6) and emotional disorders (scenario 1: adjusted OR=2.0, 95%CI: 1.2-3.3, scenario 2: OR=1.8, 95%CI: 1.1-3.1). At age 13 independent of the assumption maternal ED was not associated with offspring psychiatric disorders (scenario 1: OR=1.2, 95%CI: 0.9-1.7; scenario 2: OR=1.1, 95%CI: 0.7-1.8); and moderately associated with emotional disorders (scenario 1: OR=2.0, 95%CI: 1.2-3.2, scenario 2: OR=1.5, 95%CI: 0.7-2.9).

Hence sensitivity analyses led to similar estimates for the effect of maternal ED on the outcomes to the analyses shown in Table 2 and 3.

Additive effects

We assessed whether maternal ED and other psychiatric disorders had an additive effect on offspring any psychiatric disorder and offspring emotional disorders at age 7 by testing the significance of the interaction term between maternal ED and other psychopathology. This showed no evidence of departure from additivity on the log-odds scale (OR=1.0, p=0.9).

In relation to emotional disorders at age 7, no evidence of departure from additivity on the log-odds scale was shown for the interaction term between maternal ED and other psychopathology (OR=0.8, p=0.5) and therefore that

exposure to both of these two factors does not appear to increase/decrease the effect that they exert when present in the absence of the other.

Longitudinal effects on offspring psychiatric disorders

Because of missing covariate data these analyses involved 9,349 children, for any psychiatric disorder, and 8,316 children for emotional disorders.

These models showed an overall adjusted average effect of maternal ED on any offspring psychiatric disorder that was raised but not significant (OR=1.9, 95%CI: 0.8-4.2, p=0.1).

In contrast, for offspring emotional disorders the longitudinal random effects model showed a significant adjusted average effect of maternal ED (OR=2.3, 95%CI: 1.3-3.8, p=0.001).

Effect of maternal ED over time

In relation to any offspring psychiatric disorder, there was no effect of age on the outcome, meaning no temporal trends in incidence, and the interaction between maternal ED and age was not significant, meaning no time changing effect of the exposure.

The association between maternal ED and offspring emotional disorder across ages was also similarly tested. In this case children in the middle time-point (age 10) had increased odds of emotional disorders (OR=1.3, 95%CI: 1.1-1.7, p=0.01), controlling for exposure and confounders. However, no interaction between maternal ED and age was found, suggesting that the effect of maternal ED did not vary across time.

Discussion

This is the first longitudinal study to investigate offspring psychopathology across childhood and early adolescence in children of women with ED. We aimed to investigate the effect of maternal ED as well as other psychiatric disorders on offspring psychiatric disorders. Overall our study suggests that offspring of women with ED have higher odds of psychiatric disorders. Children of mothers with ED had a two-fold increased odds of having an emotional disorder between ages 7 and 13 years, especially anxiety disorders. Children of women who reported a prior-to-pregnancy AN (either AN or AN+BN) had higher odds of having an emotional disorder throughout childhood/early adolescence. Maternal other psychopathology prior-to-pregnancy was associated with all psychiatric outcomes across ages. There was evidence of an additive effect of maternal ED and other psychopathology on offspring psychiatric disorders and emotional disorders. However the effect of maternal ED did not seem to vary across offspring ages.

Strengths and limitations

Our findings have to be considered taking into account relevant strengths and limitations.

Firstly, due to the nature of the study (longitudinal prospective) attrition bias needs to be taken into account. Although no selective attrition in relation to maternal ED was identified, there was an over-representation of more highly educated mothers, less likely to have psychiatric disorders amongst participants. Moreover missing data on childhood psychopathology at later waves was predicted by psychopathology at earlier waves. We carried out several sensitivity analyses to assess the direction of bias, and determine if attrition was

distorting the exposure-outcome associations. Replacing the missing outcomes with values carried over from previous times did not lead to different conclusions, nor did replacing them with their opposite. Assuming missingness was at random, we also fitted random effects models and found again that the results were in line with those only including complete outcome data.

Secondly, although information on childhood psychopathology was also obtained from teachers at age 7, this was not the case at later ages. Shared method variance might therefore explain part of the association seen between maternal ED and offspring psychopathology. This might be particularly true for emotional disorders (teachers were not given the emotional disorders sections of the DAWBA, due to the known under-recognition of emotional disorders by teachers).[13] However, we carried out some sensitivity analyses (on 3,886 children) using the emotional subscale of the Strengths and Difficulties Questionnaire[18] completed by parents and teachers at age 7 and found that maternal ED was as strongly associated with emotional symptoms as rated *both* by teachers and parents, hence we suggest bias might be minimal.

Thirdly, although this is a large sample there is evidence that DAWBA computerized diagnoses lead to an underestimate of psychiatric disorders;[13] therefore the number of children across time-points who received a diagnosis was small, resulting in low power to detect associations. Indeed this is highlighted by wide confidence intervals in many or our estimates. However this might result in non-detection of true associations, rather than false positive associations.

Finally, although the mothers were asked about a range of psychiatric disorders to determine exposure, few questions about psychiatric disorders were included. We have previously validated maternal ED exposure,[15,16] and found that maternal history of depression was highly consistent with pre and postnatal depression measured by validated instruments.[19] However women were not asked about anxiety disorders. Moreover although maternal psychopathology prior to pregnancy is clearly an important indicator, we do not have information on continuing psychopathology over time (during child development) and cannot infer whether this might have played a role in offspring risk.

Despite limitations this study has several strengths; it is the first study of its nature to focus on childhood/early adolescent psychopathology. It is a longitudinal study covering 13 years of data collection. Maternal exposure was collected prior to the outcomes, allowing a prospective investigation. Although ALSPAC is not representative of the whole UK population, it is representative of its geographical area, and its nature, a community-based sample, excludes selection bias common in studies on clinical populations. The availability of data on childhood psychopathology from teachers' assessments is a great strength.

Comparison with previous findings

Increasing evidence on large population-based samples suggests that maternal psychopathology is an important risk factor for offspring psychopathology.[2,3] Both studies have highlighted low familial concordance of specific disorder, but rather a broad cross-disorder risk. Possible reasons for

non-specificity might be genetic vs. environmental transmission,[3] as well as our still-imperfect understanding and classification of psychiatric disorders.[20]

Three previous small studies investigated the effect of maternal ED on offspring psychopathology.[8-11] Our findings are consistent with these findings, and with findings from our group on this same sample highlighting increased odds of psychopathology in offspring of ED women at age 3 and ½, and from the MoBa study on the effects of maternal ED on infant emotionality and anxiety.[21,22,23]

This is the first study to investigate whether maternal ED have an additive effect with other maternal psychiatric disorders in predicting offspring disorder. Unfortunately we did not have information on paternal disorders to be able to investigate this aspect.

Our findings of a specific association between maternal ED (in particular AN) and emotional disorders is consistent with previous research highlighting a strong familial association between AN and emotional/anxiety disorders and a shared transmission of ED and anxiety disorders.[24,25] Indeed a twin study highlighted a shared genetic liability for anxiety, depression and ED.[26] Moreover there is good evidence that ED (AN in particular) are highly comorbid with anxiety disorders.[27,28] One explanation for our findings is that there is a common genetic diathesis for both emotional disorders and ED, however there have also been suggestions that AN is but a variant of anxiety disorders.[27]

There is still considerable uncertainty about aetiological pathways for ED as well as the long-term effects of maternal ED. Although this study was able to

answer some questions about familial associations between ED and psychiatric disorders in childhood/adolescence, it raises several interesting questions and pointers to future research. Firstly, although evidence on a shared liability to emotional disorders and ED is increasing, our understanding of what lies at the root of this liability remains to be determined.

Research on the long-term impact of maternal ED on the offspring is increasingly highlighting effects on psychological and eating development,[30,31] however uncertainties remain about the genetic vs. environmental nature of these effects.

The intergenerational transmission of ED has not been a focus of research, possibly due to the low overall prevalence of AN and BN in the population; large cohort studies have been recently set-up that might in the future help understand this. We have recently collected data on ED psychopathology in the ALSPAC offspring in late adolescence and will focus on intergenerational patterns of disorder-specific transmission.

Adequate prevention and early intervention strategies aimed at preventing offspring psychiatric disorder will require clarity about mechanisms of intergenerational transmission to be effective. However the role of parental mental health in predicting offspring psychopathology should be considered at a public health level, both for primary prevention as well as for targeted prevention.

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Table 1: Socio-demographic characteristics by maternal exposure

	AN (N=126)	BN (N=156)	AN+BN (N=62)	All ED (N=344)	Unexposed (N=9,099)
Child gender (male, N %)	69 (54.8%)	75(48.1%)	29(46.8%)	173 (50.3%)	4,630 (50.9%)
Child ethnicity, (Caucasian, N %)	117 (97.5%)	150 (99.3%)	60 (100%)	327 (95.1%)	8,577 (98.1)
Missing N (%)	*6 (4.8%)*	*5(3.2%)*	*2(3.2%)*	*13 (3.8%)*	*357 (3.9%)*
Maternal parity, (multiparae vs primiparae N, %)	64 (50.8%)	80 (51.3%)	29 (46.8%)	173 (50.3%)	4,868 (53.5%)
Missing N (%)	*6 (4.8%)*	*3 (1.9%)*	*2 (3.2%)*	*11(3.2%)*	*254 (2.8%)*
Maternal education at enrollment (A-level or degree vs up to GSCE)	60 (47.6%)	63 (40.4%)	37 (59.7%)	160 (46.5%)	3,325 (36.5%)
Missing N (%)	*6 (4.8%)*	*4 (2.6%)*	*2 (3.2%)*	*12 (3.5%)*	*321 (3.5%)*
Any maternal psychiatric disorder pre-pregnancy	46 (36.5%)	38 (24.4%)	33 (53.2%)	117 (34.0%)	802 (8.8%)

ED: eating disorders; AN: anorexia nervosa, BN: bulimia nervosa, OR: Odds ratio, ADHD: attention deficit hyperactivity disorder

Table 2: Psychopathology at age 7 and 10: crude and adjusted OR (95%CI) for comparison between index groups and unexposed from logistic regression

PSYCHOPATHOLOGY AT AGE 7	ALL ED (N=344)	AN (N=126)	BN (N=156)	AN+BN (N=62)
Any DSM-IV or ICD-10 disorder (N=9,443), Crude OR	1.7**(1.2-2.4)	1.7 (0.9-3.1)	1.7*(1.0-2.9)	1.7 (0.7-4.0)
Adjusted [1]	1.3 (0.9-2.0)	1.3 (0.7-2.4)	1.5 (0.8-2.5)	1.2 (0.5-2.8)
Adjusted [2]	1.4 (0.9-2.0)	1.3 (0.7-2.5)	1.5(0.9-2.6)	1.3 (0.5-3.0)
	ALL ED (N=282)	AN (N=102)	BN (N=128)	AN+BN (N=52)
Any DSM-IV or ICD-10 emotional disorder (N=7,757), Crude OR	2.7***(1.6-4.7)	3.6**(1.6-7.9)	1.2 (0.4-3.7)	5.2**(2.0-13.2)
Adjusted [1]	2.2**(1.5-3.3)	2.9** (1.3-6.4)	1.1(0.3-3.5)	3.6***(1.4-9.4)
Adjusted [2]	2.2**(1.3-3.8)	2.8**(1.3-6.3)	1.1 (0.3-3.4)	3.5**(1.3-9.3)
Any anxiety disorder, Crude OR	3.5***(2.0-6.0)	4.6***(2.1-10.0)	1.5(0.5-4.7)	6.6***(2.6-16.8)
Adjusted [1]	3.0***(1.7-5.3)	3.9**(1.8-8.8)	1.4(0.4-4.4)	5.1**(1.9-13.6)
Adjusted [2]	3.0***(1.7-5.2)	3.8**(1.7-8.6)	1.4(0.4-4.5)	5.0**(1.9-13.3)
	ED (N=256)	AN (N=93)	BN (N=111)	AN+BN (N=50)
Any DSM-IV behavioural disorder (N=9,399), Crude OR	1.1 (0.7-1.9)	0.8 (0.3-2.2)	1.5 (0.8-2.9)	0.8 (0.2-3.4)
Adjusted [1]	0.9 (0.5-1.6)	0.5 (0.2-1.7)	1.3 (0.7-2.6)	0.6(0.1-2.4)
Adjusted [2]	1.0 (0.6-1.7)	0.6 (0.2-1.8)	1.3(0.7-2.7)	1.1(0.3-3.5)
Any ADHD (N=9,411), Crude OR	1.3 (0.7-2.5)	1.4(0.5-3.9)	1.1(0.4-3.1)	1.4 (0.4-5.9)
Adjusted [1]	1.0(0.5-1.9)	1.0(0.4-2.9)	0.9(0.3-2.6)	0.9(0.2-3.8)
Adjusted [2]	1.0 (0.5-2.0)	1.1 (0.4-3.1)	1.0(0.3-2.7)	1.1 (0.3-4.6)

PSYCHOPATHOLOGY AT AGE 10 (N=7,069)	ALL ED (N=258)	AN (N=95)	BN (N=113)	AN+BN (n=50)
Any DSM-IV or ICD-10 disorder, Crude OR	1.7**(1.1-2.6)	2.4**(1.3-4.5)	1.8(1.0-3.4)	2.3*(1.0-5.5)
Adjusted [1]	1.7**(1.1-2.6)	1.9* (1.0-3.6)	1.6(0.9-3.1)	1.5 (0.6-3.7)
Adjusted [2]	1.7**(1.1-2.6)	1.9* (1.0-3.6)	1.6(0.9-3.1)	1.6 (0.6-3.8)
Any DSM-IV or ICD-10 emotional disorder, Crude OR	2.4**(1.4-4.1)	2.2 (0.8-5.3)	1.8 (0.7-4.4)	4.4**(1.7-11.2)
Adjusted [1]	1.8*(1.02-3.1)	1.5 (0.6-3.9)	1.5 (0.6-3.9)	2.6*(1.0-6.8)
Adjusted [2]	1.8*(1.04-3.2)	1.6 (0.6-4.0)	1.6 (0.6-3.9)	2.7*(1.1-7.3)
Any anxiety disorder, Crude OR	2.7**(1.5-4.9)	2.8* (1.1-7.1)	1.4 (0.4-4.5)	5.8***(2.3-14.9)
Adjusted [1]	2.1*(1.1-3.8)	2.1 (0.9-5.4)	1.2 (0.4-4.0)	3.6**(1.4-9.6)
Adjusted [2]	2.1 *(1.1-3.8)	2.2 (0.8-5.5)	1.2 (0.4-4.0)	3.8**(1.4-10.0)
	ED (N=256)	AN (N=93)	BN (N=111)	AN+BN (n=50)
Any DSM-IV behavioural disorder, Crude OR	2.0**(1.2-3.3)	2.4* (1.1-5.2)	1.7 (0.7-3.8)	1.9 (0.6-6.2)
Adjusted [1]	1.7† (1.0-2.8)	2.0(0.9-4.3)	1.5 (0.7-3.6)	1.4 (0.4-4.6)
Adjusted [2]	1.7† (1.0-2.9)	2.0 (0.9-4.3)	1.6 (0.7-3.6)	1.4 (0.4-4.6)
Any ADHD, Crude OR	1.6 (0.7-3.6)	-	2.4(0.9-6.7)	2.8 (0.7-11.6)
Adjusted [1]	1.2 (0.5-2.9)		2.2(0.8-6.0)	1.8(0.4-7.6)
Adjusted [2]	1.3 (0.5-3.0)		2.2 (0.8-6.3)	1.9 (0.4-8.2)

All groups are compared to unexposed *:p<0.05; **p≤0.01; ***:p≤0.0001; †:p=0.06;
[1]: adjusted for other psychiatric disorders
[2]: additionally adjusted for sex, parity, maternal education, ethnicity
ED: eating disorders; AN: anorexia nervosa, BN: bulimia nervosa, OR: Odds ratio, ADHD: attention deficit hyperactivity disorder

Table 3: Psychopathology and ED at age 13: Crude and adjusted OR (95%CI) for comparison between index groups and unexposed from logistic regression

PSYCHOPATHOLOGY AT AGE 13 (N=6,438)	ALL ED (N=245)	AN (N=93)	BN (N=108)	AN+BN (N=44)
Any DSM-IV or ICD-10 disorder§, Crude OR	1.4 (0.9-2.3)	1.2(0.5-2.8)	1.1(0.5-2.4)	2.9*(1.2-6.8)
Adjusted [1]	1.1 (0.7-1.9)	0.9 (0.4-2.2)	1.0 (0.4-2.2)	1.9 (0.8-4.6)
Adjusted [2]	1.2 (0.7-1.9)	1.0 (0.4-2.3)	1.0 (0.4-2.2)	2.0 (0.8-5.0)
Any DSM-IV or ICD-10 emotional disorder, Crude OR	2.1*(1.1-4.0)	1.2 (0.3-4.8)	1.5 (0.5-4.8)	5.3**(1.9-15.0)
Adjusted [1]	1.5 (0.7-3.0)	0.8 (0.2-3.4)	1.3 (0.4-4.2)	3.2*(1.1-9.3)
Adjusted [2]	1.5 (0.8-3.4)	0.8 (0.2-3.5)	1.3 (0.4-4.3)	3.1*(1.1-9.2)
Any anxiety disorder, Crude OR	2.2*(1.1-4.8)	1.6 (0.4-6.8)	1.4 (0.3-5.9)	5.5**(1.7-18.2)
Adjusted [1]	1.8 (0.8-3.6)	1.4 (0.3-5.4)	1.3 (0.3-5.4)	4.0*(1.2-13.8)
Adjusted [2]	1.8 (0.8-4.0)	1.3 (0.3-5.6)	1.3 (0.3-5.4)	3.7*(1.1-13.0)
Any DSM-IV behavioural disorder, Crude OR	0.9 (0.3-2.8)	0.9 (0.3-2.9)	1.0 (0.3-2.5)	1.6 (0.6-4.7)
Adjusted [1]	0.7(0.2-2.3)	0.7(0.2-2.4)	0.9 (0.3-2.5)	1.6 (0.6-4.7)
Adjusted [2]	0.7 (0.2-2.4)	0.7 (0.2-2.3)	0.9 (0.3-2.6)	1.9 (0.6-5.5)
Any ADHD, Crude OR	1.3 (0.7-2.4)	0.8 (0.1-5.7)	0.7 (0.1-5.3)	1.7(0.2-12.2)
Adjusted [1]	1.0 (0.5-3.7)	0.6 (0.1-4.5)	0.6 (0.1-4.4)	1.1 (0.1-8.4)
Adjusted [2]	1.0 (0.5-1.9)	0.6 (0.1-4.3)	0.7 (0.1-4.7)	1.3 (0.2-10.1)

All groups are compared to unexposed (N=6,833) *:p<0.05; **p≤0.01; ***:p≤0.0001; †:p≤0.1;
[1]: adjusted for other psychiatric disorders
[2]: additionally adjusted for sex, parity, maternal education, ethnicity
ED: eating disorders; AN: anorexia nervosa, BN: bulimia nervosa, OR: Odds ratio

Table S1: Psychopathology at age 7, 10 and 13: Frequency of clinical diagnoses

Age 7	**All ED (N=344)**	**AN (N=126)**	**BN (N=156)**	**AN+BN (N=62)**	**Unexposed (N=9,099)**
Any DSM-IV or ICD-10 disorder	33 (9.6%)	12 (9.5%)	15 (9.6%)	6 (9.7%)	536 (5.9%)
Any DSM-IV or ICD-10 emotional disorder[1]	15 (5.3%)	7 (6.9%)	3 (2.4%)	5 (9.4%)	151 (2.0%)
Any depressive disorder[1]	3 (1.1%)	2 (2.0%)	0	1 (1.9%)	44 (0.6%)
Any DSM-IV or ICD-10 anxiety disorder[1]	15 (5.3%)	7 (6.9%)	3 (2.3%)	5 (9.4%)	120 (1.6%)
Separation anxiety[1]	10 (3.6%)	4/101 (3.9%)	2/127 (1.6%)	4/53 (7.5%)	55 (0.7%)
Specific phobia[1]	5 (1.8%)	3/101 (3.0%)	1/128 (0.8%)	1/52 (1.9%)	66 (0.9%)
Social phobia[1]	1 (0.4%)	0	0	1/53 (1.9%)	8 (0.1%)
OCD[1]	0	0	0	0	3 (0.04%)
GAD[1]	1(0.4%)	0	0	1/53 (1.9%)	15 (0.2%)
Any DSM-IV behavioural disorder	15/343 (4.4%)	4 (3.2%)	9 (5.8%)	2/61 (3.3%)	353/9.056 (3.9%)
Any ADHD	10/343 (2.9%)	4/125 (3.2%)	4 (2.6%)	2 (3.2%)	204/9.056 (2.2%)
Age 10	**ALL ED (N=257)**	**AN (N=95)**	**BN (N=113)**	**AN+BN (n=50)**	**Unexposed (N=6,812)**
Any DSM-IV or ICD-10 disorder	29 (11.3%)	12 (12.6%)	11 (9.7%)	6 (12.2%)	385 (5.6%)
Any emotional disorder	15(5.8%)	5 (5.3%)	5 (4.4%)	5 (10.2%)	172 (2.5%)
Any depressive disorder	4 (1.6%)	0	2 (1.9%)	2 (4.3%)	57 (0.9%)
Any anxiety disorder	13 (5.1%)	5 (5.3%)	3 (2.7%)	5 (10.2%)	130 (1.9%)
Separation anxiety	8/250 (3.2%)	5/92(5.4%)	2/111(1.8%)	1/48 (2.1%)	63/6,555 (1%)
Specific phobia	5/255 (2.0%)	1/93 (1.1%)	1 (0.9%)	3/49 (6.1%)	50/6,781 (0.7%)
Social phobia	0	0	0	0	23/6,791 (0.3%)
OCD	0	0	0	0	4/6,795 (0.06%)
GAD	1/255 (0.4%)	0	0	1/49 (2.0%)	26/6,769 (0.4%)
Any behavioural disorder	16/255 (6.3%)	7/94 (7.5%)	6/112 (5.4%)	3/49 (6.1%)	222/6,758 (3.3%)
Any ADHD	6/255 (2.3%)	0	4/112 (3.6%)	2/49 (4.1%)	102/6,784 (1.5%)

Age 13	**All ED (N=245)**	**AN (N=93)**	**BN (N=108)**	**AN+BN (n=44)**	**Unexposed (N=6,193)**
Any DSM-IV disorder	18 (7.3%)	6 (6.5%)	6 (5.6%)	6 (13.3%)	327 (5.2%)
Any emotional disorder	9 (3.7%)	2 (2.2%)	3 (2.8%)	4 (8.9%)	116(1.9%)
Any depressive disorder	5 (2.1%)	0/89	3/106 (2.8%)	2 (4.4%)	48/6,071 (0.8%)
Any anxiety disorder	7 (2.8%)	2 (2.2%)	2 (1.9%)	3(6.7%)	82 (1.3%)
Separation anxiety	0/224	0/86	0/99	0/39	33/5,689 (0.5%)
Specific phobia	4/243 (1.6%)	1/91 (1.1%)	1 (0.9%)	2 (4.4%)	25 (0.4%)
Social phobia	4(1.6%)	2 (2.2%)	1 (0.9%)	1 (2.2%)	22/6,171 (0.3%)
OCD	1/243 (0.4%)	0/92	0/107	1 (2.2%)	6/6,172 (0.1%)
GAD	1/244 (0.4%)	0/92	0	1 (2.2%)	25/6,157 (0.4%)
Any behavioural disorder	11/241 (4.5%)	3/90 (3.3%)	4/107 (3.7%)	4 (8.9%)	223/6,148 (3.6%)
Any ADHD (DSM-IV)	3/243 (1.2%)	1/92 (1.1%)	1/107(0.9%)	1 (2.2%)	85/6,165 (1.4%)

[1]Data on emotional disorders at age 7 were available from parents only (N=7,757)

ED: eating disorders; AN: anorexia nervosa, BN: bulimia nervosa, OR: Odds ratio, ADHD: attention deficit hyperactivity disorder, OCD: obsessive compulsive disorder, GAD: generalized anxiety disorder

Supplemental Table 2: Maternal other pre-pregnancy psychopathology and offspring psychopathology at ages 7, 10, 13: crude and adjusted OR (odds ratios) and 95% Confidence intervals for comparisons with unexposed

PSYCHOPATHOLOGY	Age 7		Age 10		Age 13	
	N	Other maternal psychopathology (N=919)	N	Other maternal psychopathology (N=630)	N	Other maternal psychopathology (N=656)
Any DSM-IV or ICD-10 disorder, Crude OR	9,443	2.2***(1.8-2.8)	7,069	2.5***(1.9-3.3)	6,438	2.4***(1.8-3.2)
Adjusted [1]		2.2***(1.7-2.7)		2.4***(1.8-3.1)		2.3***(1.7-3.2)
Adjusted [2]		2.2***(1.7-2.7)		2.5***(1.9-3.2)		2.3***(1.7-3.2)
		N=710		N=630		N=565
Any DSM-IV or ICD-10 emotional disorder, Crude OR	7,757	2.4***(1.6-3.6)	7,069	3.2***(2.2-4.5)	6,438	3.0***(1.9-4.6)
Adjusted [1]		2.2***(1.5-3.3)		3.0***(2.1-4.2)		2.8***(1.8-4.4)
Adjusted [2]		2.2***(1.5-3.4)		3.0***(2.1-4.2)		2.8***(1.8-4.5)
Any anxiety disorder, Crude OR	7,757	2.1**(1.3-3.3)		3.0***(2.0-4.6)		2.2**(1.2-3.8)
Adjusted [1]		1.8*(1.1-2.8)		2.8***(1.8-4.2)		1.9*(1.1-3.5)
Adjusted [2]		1.8*(1.1-2.9)		2.8***(1.8-4.2)		2.0*(1.1-3.6)
		N=710		N=617		N=555
Any DSM-IV behavioural disorder, Crude OR	9,399	2.0***(1.5-2.7)	7,013	2.1***(1.5-3.0)	6,389	2.6***(1.9-3.7)
Adjusted [1]		2.0***(1.5-2.7)		2.0***(1.3-2.8)		2.2**(1.3-3.9)
Adjusted [2]		2.0***(1.5-2.7)		2.0***(1.4-2.8)		2.2**(1.3-3.9)
Any ADHD, Crude OR	9,411	2.5***(1.8-3.5)	7,013	2.1***(1.5-3.0)	6,389	2.6***(1.9-3.7)
Adjusted [1]		2.5***(1.7-3.5)		2.5***(1.5-4.1)		2.6***(1.9-3.7)
Adjusted [2]		2.4***(1.7-3.5)		2.4***(1.5-4.0)		2.6***(1.8-3.6)

All groups are compared to unexposed *:p<0.05; **p≤0.01; ***:p≤0.0001;
[1]: adjusted for maternal eating disorders; [2]: additionally adjusted for sex, parity, maternal education, ethnicity

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